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## FIT Clinical Decision Making

## LOFFLER'S ENDOCARDITIS PRESENTING AS CARDIOGENIC SHOCK SECONDARY TO SEVERE SYSTOLIC AND DIASTOLIC BIVENTRICULAR FAILURE

Moderated Poster Contributions

Pulmonary Hypertension and FIT Clinical Decision Making Moderated Poster Theater, Poster Hall B1

Saturday, March 14, 2015, 11:00 a.m.-11:10 a.m.

Session Title: FIT Clinical Decision Making: Moderated Poster Session I

Abstract Category: Heart Failure and Cardiomyopathies

Presentation Number: 1127M-11

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**Background:** Cardiac manifestation of hypereosinophilic syndrome present as Löffler's endocarditis (LE) and can lead to endomyocardial disease characterized by restrictive cardiomyopathy due to endomyocardial fibrotic thickening and biventricular thrombus formation.

**Case:** 46 year old female with history of asthma and allergies to multiple medications presented with 1 week history of dyspnea, orthopnea, weight gain and fatigue. Physical examination revealed signs of cardiogenic shock; elevated jugular venous pressure, peripheral edema, pulmonary rales, cold and mottled extremities and hypotension. Laboratory values showed eosinophilia ( $1150/\mu\text{L}$ ), acute kidney injury and lactic acidosis.

**Decision Making:** Echocardiogram and cardiac MRI showed restrictive filling, severe left ventricular (LV) systolic dysfunction and large biventricular apical thrombi. Intra aortic balloon pump was placed; anticoagulation and inotropes initiated. Coronary angiogram showed normal coronaries. Clinical index of suspicion was high for LE. Work up was negative for secondary causes of eosinophilia. Endomyocardial biopsy (EB) was not pursued due to high risk of cardioembolism. Bone marrow biopsy showed extensive eosinophilic infiltration. Methylprednisolone was started. Her cardiogenic shock resolved and goal directed medical therapy for cardiomyopathy begun. Echocardiogram a month later showed improved LV function, shrinkage of the LV thrombus and resolution of the right ventricular thrombus. EB was done then and showed interstitial edema and lymphocytic infiltrate without eosinophils; staining was positive for eosinophil granule protein (EGP) consistent with LE. She became eligible for mepolizumab, a steroid sparing agent and continued to clinically improve. The gene mutation that predicts response to imatinib was absent.

**Conclusion:** Presence of biventricular apical thrombi with restrictive filling vis characteristic of LE. Eosinophilia may be mild or absent at presentation. EB may not show eosinophilic infiltration but stains positive for EGP; fibrosis may be absent in early stages. Steroids are mainstay of treatment. Other options include imatinib, hydroxyurea and mepolizumab.